ANNALS OF SURGERY
Vol. 233, No. 2, 176–182


# Central and Peripheral Adverse Hemodynamic Changes During Laparoscopic Surgery and Their Reversal with a Novel Intermittent Sequential Pneumatic Compression Device

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**Objective**

To study the influence of a novel intermittent sequential pneumatic compression device (Lympha-press) on the adverse cardiac and peripheral hemodynamic changes induced by positive-pressure pneumoperitoneum (PPPn) in laparoscopic surgery.

**Summary Background Data**

Creation of PPPn is known to cause adverse central and peripheral hemodynamic changes. An intrasubject observational study was undertaken to quantitate these adverse changes and to assess the influence of an intermittent sequential pneumatic compression system on these adverse hemodynamic changes during laparoscopic surgery with PPPn.

**Methods**

The study involved 16 consecutive patients undergoing laparoscopic surgery with PPPn of 12 mmHg and 30° head-up tilt position. The following peripheral hemodynamic recordings were made using Doppler ultrasound: peak systolic velocity (PSV), end diastolic velocity (EDV), and cross-sectional area of the femoral vein. Central monitoring included cardiac output and stroke volume by transesophageal Doppler, blood pressure, and pulse. The hemodynamic state based on these parameters was assessed before induction of PPPn with the anesthetized patient in the supine position, after induction of PPPn and head-up tilt position with Lympha-press off, and during PPPn and head-up tilt position with Lympha-press on, and after desufflation with the patient in the supine position under general anesthesia.

**Results**

Positive-pressure pneumoperitoneum and the head-up tilt position resulted in a 33% reduction in PSV, a 21% reduction in EDV, and a 29% increase in cross-sectional area of the femoral vein. This was associated with a 20% reduction in cardiac output and an 18% reduction in stroke volume. Activation of Lympha-press during PPPn and the head-up tilt position resulted in a 129% increase in PSV and a 55% increase in EDV by 55%. It also increased the cardiac output by 27% and stroke volume by 16%, with no effect on cross-sectional area. Compared with the pre-PPPn stage, there was no difference in cardiac output or stroke volume, but the PSV was higher by 78% and the EDV by 32%. After abdominal desufflation in the supine position, the cardiac output and stroke volume were restored to the pre-PPPn level, but persistent and significant elevations were observed during the period of study in PSV, EDV, and cross-sectional area.

**Conclusions**

Significant and individually variable central and peripheral hemodynamic changes are encountered during laparoscopic surgery with PPPn and the head-up tilt position. These are reversed by intermittent sequential pneumatic compression using Lympha-press.

Compared with open surgery, laparoscopic surgical procedures are associated with reduced traumatic insult and metabolic stress to the patient and hence a smoother postoperative period and accelerated recovery.[1,2] On these grounds, the laparoscopic approach has been proposed for surgery on high-risk patients with comorbid cardiopulmonary disease.[3] However, there are documented adverse cardiovascular, hormonal, and neuroendocrine changes[1,4,5] caused by positive-pressure pneumoperitoneum (PPPn), and there have been reports of sudden intraoperative cardiovascular collapse or severe pulmonary edema requiring ventilation after uneventful laparoscopic cholecystectomy.[6,7]

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Accepted for publication May 12, 2000.

The adverse effects on cardiac performance produced by PPPn may increase the risk of cardiac complications in susceptible patient groups.[8–10] In addition to the central changes, PPPn results in reduced peripheral venous flow[11–16] and diminished perfusion of intraabdominal organs.[17–19]

In addressing this problem, several remedies have been proposed as contenders for clinical evaluation.[20] One of these is a mechanical solution that involves the application of intermittent sequential pneumatic compression (ISPC) of the lower limbs during laparoscopic surgery. The beneficial effects of ISPC devices on the peripheral venous flow induced by PPPn have been documented.[15,16,21] The present study was designed to evaluate the effect of a novel ISPC system (Lympha-press, Mego Afek Kibbutz afek 30042, Israel) on both the central and peripheral hemodynamic changes induced by PPPn during laparoscopic surgery.

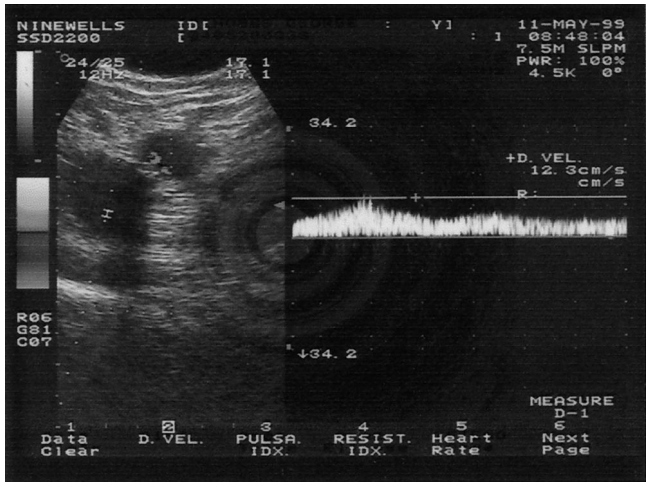


**Figure 1.** Combined femoral venous velocity profile at center of femoral vein and cross sectional area are shown in Duplex (B and D) mode. Peak systolic velocity (PSV) of femoral vein is measured at 12.3 cm/sec.

## METHODS

Informed consent was secured from 16 patients in American Surgical Association (ASA) categories I and II (5 men ages 26–65 years, 11 women ages 38–78 years) who were undergoing laparoscopic surgery (cholecystectomy, fundoplication, liver thermal ablation) at Ninewells Hospital, Dundee, Scotland. The prospective study was designed as a paired investigation, with each patient acting as his or her own control. Ethical approval was obtained from the Tayside Committee on Medical Research Ethics. The exclusion criteria were previous pulmonary embolism and deep vein thrombosis, abnormal coagulopathy, chronic venous insufficiency, and ASA categories III and IV. Only three patients were in ASA category II, one of whom had moderate stable angina and a history of myocardial infarction.

### Peripheral Venous Flow Studies

Femoral venous cross-sectional area and velocities were obtained using an ultrasound Doppler machine (Aloka Diagnostic System, SSD-2200, Mitaka-Shi, Tokyo, Japan). The technique involved identifying the bifurcation of the profunda femoris artery from the common femoral artery and then selecting a segment of femoral vein just proximal to this area. The window of the linear array ultrasound microconvex probe (7.5 MHz) (Aloka, UST-995) was secured in a customized template fixed at 45° to flow axis throughout the procedure (Fig. 1).

### Cardiac Function

Cardiac output and stroke volume were measured using a transesophageal Doppler machine (ODMII, S/N 2060, Abbott, Maidenhead, Kent, UK) with single-use 4-MHz sterile probes (Abbott Single Patient Probe G975). Blood pressure and pulse rate were recorded from an anesthetic monitoring system (M1205 A, Omnicare, Model 24/24C, Hewlett-Packard, Palo Alto, CA).

### Study Endpoints

Endpoints were peak systolic velocity (PSV) and end diastolic velocity (EDV) of femoral venous flow (cm/s), cross-sectional area of the femoral vein ($mm^2$), cardiac output (L/min), stroke volume (mL), systolic and diastolic blood pressure (mmHg), and pulse rate. All these were obtained at following stages:

- Pre-PPPn in supine position under general anesthesia with the Lympha-press off (stage 1)
- PPPn and head-up tilt position with the Lympha-press

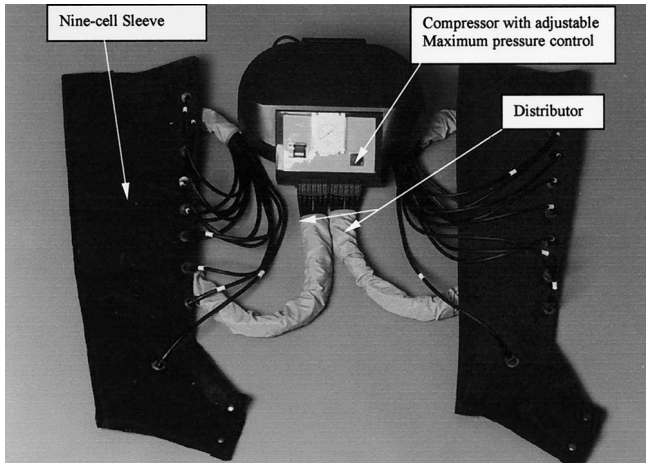


**Figure 2.** Lympha-press system, showing compressor, distributor, and multicell trousers. The pressure is distributed into overlapping air compartments from distal to proximal. Each 30-second cycle consists of 18 seconds compression and 12 seconds decompression.

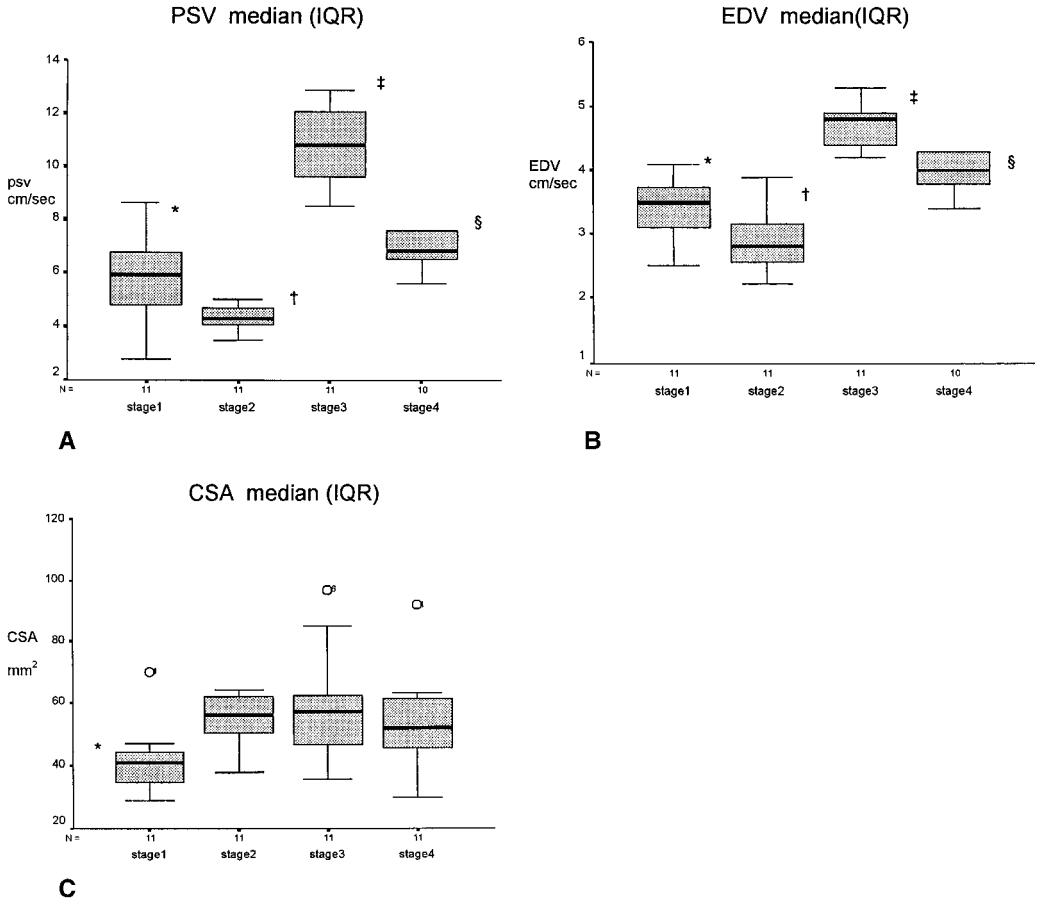


**Figure 3.** (A) Peak systolic velocity (PSV), (B) end diastolic velocity (EDV) and (C) Cross-sectional area (CSA) of femoral vein during stages 1 through 4. Stage 1 = Pre.PPPn + supine; stage 2 = PPPn + Tilt + Lympha-press, off; stage 3 = PPPn + Tilt + Lympha-press, on; stage 4 = post PPPn + supine. **P* < .01, stage 1 vs. 2; †*P* < .01, stage 2 vs. 3; ‡*P* < .01, stage 3 vs. 1; §*P* < .01, stage 4 vs. 1. **P* < .05, stage 1 vs. 4.

off (stage 2)
- PPPn and head-up tilt position with the Lympha-press on (stage 3)
- Post-PPPn in supine position under general anesthesia with the Lympha-press off (stage 4)

Femoral venous recordings were taken every 5 minutes and cardiac recordings every 2 to 3 minutes. Each recording was an average of three readings. A 5-minute stabilization time was allowed between recordings of an event (defined as a change between any two successive stages).

## Intermittent Sequential Pneumatic Compression Device

The Lympha-press system consists of a compressor, a distributor, and a multicell sleeve (Fig. 2). Each sleeve is a carpetlike wrapping of nine cells that is adapted to each lower limb from ankle to groin. The adjustable pressure generated by the pneumatic compressor is set at a maximum of 55 mmHg and is distributed into overlapping air compartments sequentially from distal to proximal to both legs simultaneously. This effect translates to a milking mechanism on the lower limb veins. The 30-second cycle consists of 18 seconds of compression (each cell is inflated for approximately 2 seconds) and 12 seconds of decompression, starting with a few seconds of intermission period when all cells are in a simultaneous compression state, followed by simultaneous decompression of all cells.

## Anesthesia and Pneumoperitoneum

A standardized general anesthetic protocol administered by two anesthetists was followed in all patients. All procedures were carried out under 12 mmHg PPPn and a 30° head-up tilt position. Patients were allowed to eat or drink up to midnight before surgery and then were fully fasted. This regimen was adjusted if patients were scheduled for a later time on the surgery list. Intraoperative fluid maintenance was provided by crystalloid infusion (7 mL/kg per hour), and extra losses were replaced as clinically indicated. End tidal $CO_2$ was maintained at 4.5 to 5.0 KPa with the ventilatory technique.

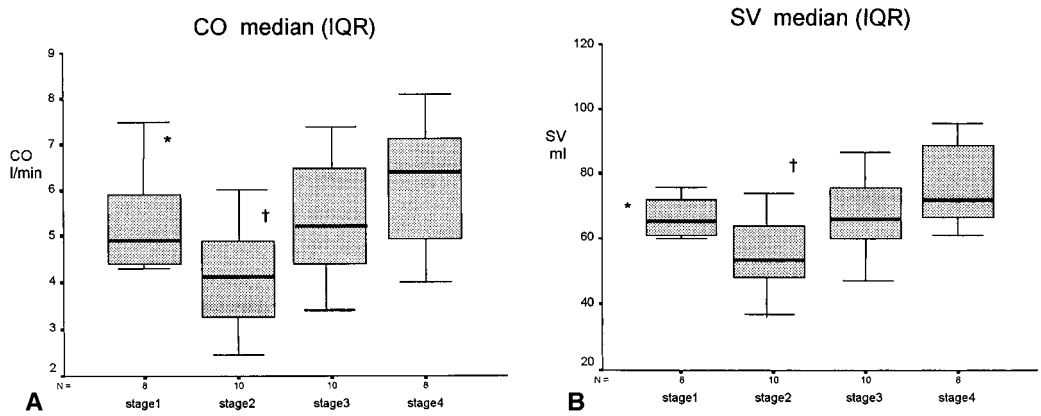


**Figure 4.** (A) Cardiac output (CO) and (B) stroke volume (SV) during stages 1 through 4. Stage 1 = Pre.PPPn + supine; stage 2 = PPPn + Tilt + Lympha-press, off; stage 3 = PPPn + Tilt + Lympha-press, on; stage 4 = post PPPn + supine. *$P < .05$, stage 1 vs. 2; †$P = .007$, stage 2 vs. 3.

## Statistical Analysis

The mean difference of outcome measures during any event was not normally distributed. Therefore, the mean difference was compared with zero as the null hypothesis in a paired analysis with the Wilcoxon rank signed test for repeated measurements using the Windows-based SPSS version 9.0 statistical program (SPSS Inc., Chicago, IL). Results are presented as median percentage change and interquartile range (25th to 75 percentiles). Significance was set at the 5% level.

## RESULTS

For technical reasons, it was possible to obtain both central and peripheral readings in only five patients. Another five patients had the central readings (cardiac output, stroke volume, blood pressure, pulse rate) only, and six patients had the peripheral readings (PSV, EDV, cross-sectional area) only. No perioperative cardiovascular complications were observed during this study. The median values of PSV, EDV, cross-sectional area, cardiac output, and stroke volume plotted against the four stages of the procedure are shown in Figures 3 and 4.

### Stage 1

The changes in cardiac output and stroke volume showed great individual variations. Overall, we observed a 20% reduction in cardiac output and an 18% reduction in stroke volume. No significant change was observed in the systolic and diastolic blood pressure or pulse rate. The peripheral changes observed were a median reduction in PSV by 33% and in EDV by 21%. The cross-sectional area of the femoral vein increased by 29% (Table 1).

### Stage 2

The Lympha-press augmented PSV by 129% and EDV by 55% but did not alter significantly the femoral vein cross-sectional area. For the 11 patients who had peripheral recordings, the augmented femoral venous flow velocity translated to a median increase of 6.2 cm/s for PSV and 1.6 cm/s for EDV (Table 2). ISPC generated by the Lympha-

**Table 1. CHANGES IN CENTRAL AND PERIPHERAL MEASUREMENTS**

| | PSV | EDV | CSA | CO | SV |
|---|---|---|---|---|---|
| Stage 1 vs. 2 | −33 (−22, −40) | −21 (−33, −15) | 29 (1, 33) | −20 (−37, −4) | −18 (−23, −8) |
| *P* value | .003 | .003 | .013 | .037 | .022 |
| Stage 2 vs. 3 | 129 (112, 194) | 55 (43, 95) | −7 (−15, 3) | 27 (17, 31) | 16 (10, 34) |
| *P* value | .003 | .003 | .213 | .007 | .007 |
| Stage 1 vs. 3 | 78 (69, 131) | 32 (17, 51) | 17 (4, 63) | −5 (−19, 15) | −5 (−12, 14) |
| *P* value | .003 | .003 | .037 | .575 | .799 |
| Stage 4 vs. 1 | 35 (10, 50) | 20 (0, 39) | 9 (6, 42) | 5 (−8, 23) | −1 (−9, 23) |
| *P* value | .006 | .028 | .038 | .753 | .463 |

Data are given as median percentage change (interquartile range).
PSV, peak systolic velocity of femoral vein; EDV, end diastolic velocity of femoral vein; CSA, cross-sectional area of femoral vein; CO, cardiac output; SV, stroke volume.

**Table 2. MEAN DIFFERENCE IN CENTRAL AND PERIPHERAL MEASUREMENTS DURING PPPN AND HEAD-UP TILT POSITION BETWEEN LYMPHA-PRESS ON OR OFF**

| Patient | PSV (cm/s) | EDV (cm/s) | CSA ($mm^2$) | CO (mL/min) | SV (mL) |
|---|---|---|---|---|---|
| 1 | 5.4 | 1.7 | 2.0 | 650 | 7.5 |
| 2 | 6.6 | 1.6 | −5.5 | 767 | 8.7 |
| 3 | 4.6 | 1.0 | −7.5 | 851 | 9.1 |
| 4 | 4.2 | 1.6 | −9.7 | −560 | −2.4 |
| 5 | 6.1 | 1.6 | −4.5 | 1,291 | 7.4 |
| 6 | | | | 1,405 | 26.9 |
| 7 | | | | 525 | 1.8 |
| 8 | | | | 958 | 8.8 |
| 9 | | | | 1,367 | 21.3 |
| 10 | | | | 2,417 | 19.2 |
| 11 | 8.9 | 2.3 | 6.4 | | |
| 12 | 7.3 | 2.6 | −16.5 | | |
| 13 | 8.9 | 1.6 | −2.1 | | |
| 14 | 5.1 | 1.4 | −3.3 | | |
| 15 | 6.3 | 2.8 | 12.0 | | |
| 16 | 3.3 | 1.1 | 0.3 | | |
| Median | 6.2 | 1.6 | −3.9 | 905 | 8.8 |
| Interquartile range | (4.5, 7.7) | (1.3, 2.4) | (−8, 2) | (619, 1,377) | (6, 20) |
| *P* value | .003 | .003 | .248 | .009 | .009 |

PSV, peak systolic velocity of femoral vein; EDV, end diastolic velocity of femoral vein; CSA, cross-sectional area of femoral vein; CO, cardiac output; SV, stroke volume.

press augmented cardiac output by 27% and stroke volume by 16% (i.e., a median increase of 0.905 L/min in cardiac output and 8.8 mL in SV). In addition, Lympha-press activation resulted in a significant elevation of both systolic and diastolic blood pressure, but it had no effect on pulse rate (Table 3).

## Stage 3

Lympha-press augmented PSV and EDV by 78% and 32%, respectively, compared with stage 1. There was also a 17% increase in the femoral vein cross-sectional area. There was no net effect on cardiac output, stroke volume, blood pressure, or pulse rate.

**Table 3. CHANGES IN BLOOD PRESSURE AND PULSE RATE**

| | Systolic | Diastolic | Pulse Rate |
|---|---|---|---|
| Stage 1 vs. 2 | 2 (−28, 17) | 2 (−13, 32) | −1 (−6, 0) |
| *P* value | .878 | .508 | .173 |
| Stage 2 vs. 3 | 19 (7, 23) | 11 (7, 16) | 4 (−3, 15) |
| *P* value | .005 | .005 | .110 |
| Stage 1 vs. 3 | 14 (−3, 38) | 17 (−7, 40) | 5 (−6, 13) |
| *P* value | .093 | .059 | .333 |
| Stage 4 vs. 1 | 7 (0, 58) | −2 (−7, 20) | 0 (−9, 30) |
| *P* value | .173 | .893 | .917 |

Data are given as median percentage change (interquartile range).

## Stage 4

Compared with stage 1, all the peripheral parameters increased with desufflation of the pneumoperitoneum and restoration of the supine position at the end of the procedure. Although the median values for cardiac output and stroke volume during stage 4 were greater than the corresponding values during stage 1, the differences were small and not significant. Similarly, no overall change was observed in either blood pressure or pulse rate.

# DISCUSSION

Our results showed a significant interpatient variability in the reduction of cardiac output and stroke volume with PPPn, as reported previously.[9,22] Thus, in one patient, a52% reduction in cardiac output was observed. It could be argued that a drop in cardiac output of this magnitude could have serious consequences in patients with risk factors for perioperative cardiac complications.[8,10,23–25] The absence of any perioperative cardiac complications in the present study is probably explained by the small cohort and the exclusions of all patients in ASA categories III and IV.

The issue of cardiac performance in the presence of a significant PPPn remains unresolved. Some have reported no change in cardiac output with the creation of PPPn;[26–28] others have suggested a dual response to a graduated elevation in the intraperitoneal pressure, with initial stimulation and subsequent suppression of CO.[5,29] Most reported studies document a variable degree of "cardiac suppression," in agreement with our findings.[7,9,24,30–36] To some extent, the reduction in cardiac output depends on the level of intraperi-

toneal pressure.[31,37] However, as demonstrated by our findings, even when the intraperitoneal pressure is kept at a constant level of 12 mmHg, the effect on cardiac output varies from patient to patient. Undoubtedly, the cardiac suppression induced by PPPn appears to be well tolerated by relatively young fit patients.[5] However, the situation is likely to be different in patients at risk of perioperative cardiac complications.[8,9,25] These cardiac changes were not accompanied by any significant changes in blood pressure and pulse rate, and thus these indices cannot be relied on to monitor cardiac changes induced by PPPn.

The validity of transesophageal Doppler measurement of cardiac function is well established and its accuracy has been confirmed against the thermodilution and Fick methods.[34] It is ideal for noninvasive monitoring of patients at risk for acute hemodynamic changes that may not be readily revealed by conventional intraoperative monitoring techniques, and its use in laparoscopic surgery for this purpose has been recommended.[9,35] The technique is safe, noninvasive, and easy to deploy, it produces little artifact, and correct interpretation does not require specialist training.[36]

The reported augmentation of venous flow generated by other ISPC devices is less than that obtained with the Lympha-press.[14–16,21] The degree of femoral venous flow augmentation with ISPC systems depends on several factors, such as the ratio of the duration of compression to decompression, the rate of insufflation, the number of cells, the distribution of pressure over the lower limbs, the maximum pressure reached in each cell, and the arrangement of the cells (i.e., overlapping vs. nonoverlapping cells). The specifications of the Lympha-press differ from those of other ISPC systems in almost all these parameters. No comparative data with other ISPC systems are available.

Some studies have linked the adverse influence of PPPn on cardiac performance to the suppression of venous return (cardiac preload) as the primary cause;[7,24] others have incriminated the significant increase in systemic vascular resistance (afterload) as the dominant factor.[1,21,25,33] In the present study, PPPn and the head-up tilt position resulted in an overall 33% reduction in PSV and a 20% reduction in cardiac output. The Lympha-press caused a 129% elevation in PSV but generated only a 27% increase in cardiac output. These data suggest that the reduction in cardiac output with the establishment of PPPn and the head-up tilt position cannot be entirely explained by reduction in venous return. It appears that both factors may be involved. The augmented venous return induced by the Lympha-press is partially dissipated in the pulmonary vasculature before influencing the cardiac output.

A variable but significant reduction of blood flow in the renal, hepatic, gastric, and mesenteric beds has been reported during laparoscopic surgery with PPPn,[17–19] and impaired renal function has been documented. The reduced tissue perfusion is probably multifactorial—loco-regional vascular changes in addition to reduced cardiac output. Thus, it is by no means certain that the increased cardiac output resulting from use of the Lympha-press would improve tissue perfusion. Further studies are needed to address this issue. Our findings demonstrate that ISPC by the Lympha-press fully reverses the cardiac depression and head-up tilt position and overcompensates for the state of peripheral venous stasis induced by PPPn during laparoscopic surgery. Application of Lympha-press is recommended, particularly for patients at risk for perioperative cardiac complications.

## References


1. Barnes GE, Laine GA, Giam PY, et al. Cardiovascular response to elevation of intrabdominal hydrostatic pressure. Am J Physiol 1985; 248:R208–213.
2. Goodale RL, Beebe DS, McNevin MP, et al. Haemodynamic, respiratory, and metabolic effects of laparoscopic cholecystectomy. Am J Surg 1993; 166:533–537.
3. Dubois F, Berthelot G, Levard H. Laparoscopic cholecystectomy: historical perspective and personal experience. Surg Laparosc Endosc 1991; 1:52–57.
4. Punnonen R, Viinäki O. Vasopressin release during laparoscopy: role of increased intra-abdominal pressure. Lancet 1982; 8264:175–176.
5. Kelman GR, Swapp GH, Smith I, et al. Cardiac output and arterial blood-gas tension during laparoscopy. Br J Anaesth 1972; 44:1155–1161.
6. Brantly JC, Riley PM. Cardiovascular collapse during laparoscopy: a report of two cases. Am J Obstet Gynecol 1998; 159:735–737.
7. Westerband A, Van De Water JM, Amzallag M, et al. Cardiovascular changes during laparoscopic cholecystectomy. Surg Gynecol Obstet 1992; 175:535–538.
8. Feig BW, Berger DH, Dupuis JF, et al. Hemodynamic effects of $CO_2$ abdominal insufflation (CAI) during laparoscopy in high-risk patients. Anesth Analg 1994;78:S1-S503.
9. Haxby J, Gray MR, Rodriguez C, et al. Assessment of cardiovascular changes during laparoscopic hernia repair using oesophageal Doppler. Br J Anaesth 1997; 78:515–519.
10. Volpino P, Cangemi V, D'Andrea N, et al. Hemodynamic and pulmonary changes during and after laparoscopic cholecystectomy. Surg Endosc 1998; 12:119–123.
11. Ido K, Suzuki T, Kimura K, et al. Lower-extremity venous stasis during laparoscopic cholecystectomy as assessed using color Doppler ultrasound. Surg Endosc 1995; 9:310–313.
12. Janseen H, Treviño C, Williams D. Hemodynamic alterations in venous blood flow produced by external pneumatic compression. J Cardiovasc Surg 1993; 34:441–447.
13. Jorgensen JO, Gillies RB, Lalak NJ, et al. Lower limb venous hemodynamics during laparoscopy: an animal study. Surg Laparosc Endosc 1991; 4:32–35.
14. Jorgensen JO, Lalak NJ, North L, et al. Venous stasis during laparoscopic cholecystectomy. Surg Laparosc Endosc 1994; 4:128–133.
15. Millard JA, Hill BB, Cook PS, et al. Intermittent sequential pneumatic compression in prevention of venous stasis associated with pneumoperitoneum during laparoscopic cholecystectomy. Arch Surg 1993; 128:914–919.
16. Schwenk W, Böhn B, Fügener A, et al. Intermittent pneumatic sequential compression (ISC) of the lower extremities prevents venous stasis during laparoscopic cholecystectomy. Surg Endosc 1998; 12:7–1.
17. Ishizaki Y, Bandai Y, Shimomura K, et al. Changes in splanchnic blood flow and cardiovascular effects following peritoneal insufflation of carbon dioxide. Surg Endosc 1993; 7:420–423.
18. Jakimowicz J, Stultiëns G, Smulders F. Laparoscopic insufflation of abdomen reduces portal venous flow. Surg Endosc 1998; 12:129–132.
19. Junghans T, Böhm B, Gründel K, et al. Does pneumoperitoneum with different gases, body positions, and intraperitoneal pressure influence renal and hepatic blood flow? Surgery 1997; 121:206–210.
20. Cuschieri A. Adverse cardiovascular changes induced by positive pressure pneumoperitoneum [editorial]. Surg Endosc 1998; 12:93–94.

21. Christen Y, Reymond MA, Vogel JJ, et al. Hemodynamics of intermittent pneumatic compression of the lower limb during laparoscopic cholecystectomy. Am J Surg 1995; 17:395–398.
22. Wittgen CM, Andrus CH, Fitzgerald SD, et al. Analysis of the hemodynamic and ventilatory effects of laparoscopic cholecystectomy. Arch Surg 1991; 126:997–1001.
23. Guidelines for perioperative cardiovascular evaluation for noncardiac surgery. Circulation 1996; 93:1278–1317.
24. Gebhardt H, Bautz A, Ross M, et al. Pathophysiological and clinical aspects of the $CO_2$ pneumoperitoneum. Surg Endosc 1997; 11:864–867.
25. Safran DB, Orlando R. Physiologic effects of pneumoperitoneum. Am J Surg 1994; 167:281–286.
26. Lindberg F, Bergqvist D, Rasmussen I, Haglund U. Hemodynamic changes in the inferior caval vein during pneumoperitoneum. Surg Endosc 1997; 11:431–437.
27. Kubota K, Kajiura N, Teruya M, et al. Alterations in respiratory function and hemodynamics during laparoscopic cholecystectomy under pneumoperitoneum. Surg Endosc 1993; 7:500–504.
28. Motew M, Ivankovich A, Bieniarz J, et al. A Cardiovascular effects and acid–base and blood gas changes during laparoscopy. Am J Obstet Gynecol 1973; 115:1002–1012.
29. Ivankovich AD, Miletich DJ, Albrecht RF, et al. Cardiovascular effects of intraperitoneal insufflation with carbon dioxide and nitrous oxide in dogs. Anesthesiology 1975; 42:281–287.
30. Lenz RJ, Thomas TA, Wilkins DG. Cardiovascular changes during laparoscopy. Anesthesia 1976; 31:4–12.
31. Williams MD, Murr PC. Laparoscopic insufflation of the abdomen depresses cardiopulmonary function. Surg Endosc 1993, 7:12–16.
32. Mckenzie R, Wadhwa RK, Bedger RC. Noninvasive measurement of cardiac output during laparoscopy. J Reprod Med 1980; 24:247–250.
33. Shuto K, Kitano S, Yoshida T, et al. Hemodynamic and arterial blood gas changes during $CO_2$ and helium pneumoperitoneum in pigs. Surg Endosc 1995; 9:1173–1178.
34. Cuschieri J, Rivers M, Cruso J, et al. Transoesophageal Doppler thermodilution and Fick cardiac output measurements in critically ill patients. Crit Care Med 1998; 26(suppl 1):A62.
35. Elliot S, Savil P, Eckersall S. Cardiovascular changes during LC: a study using transoesophageal Doppler monitoring. Eur J Anaesthesiol 1998; 15(1):50–55.
36. Gan TJ, Arrowsmith J. The oesophageal Doppler monitor, a safe means of monitoring the circulation [editorial]. Br Med J 1997; 315:893–894.
37. Ishizaki Y, Bandai Y, Shimomura K, et al. Safe intra-abdominal pressure of carbon dioxide pneumoperitoneum during laparoscopic surgery. Surgery 1993; 114:549–554.